Heretofore Unrecognized Type of Pain Signals from Large Colon May Form Common Link Between Belligerent Behavior Associated with Consumption of Brown Liquors, Colic-Associated Dystemperament, Violence in ASD and Irritability in PMS

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Introduction

At a time when the majority of research into the intestine-brain dynamic is centered around the emergent fields of microbiomics and the notion that many neurological symptoms may have an inflammatory trigger, this author would take the opportunity to submit for public consideration that the aforementioned trends in research may be, at times, having the effect of slowing medical progress by tempting doctors in both practical and research settings to attribute nearly all aspects of medical phenomena under the nebulous aegis of the so-called “Brain-Gut Connection” to these previously overlooked biological features.

Abstract

At present, the latest popularly-promoted hypothesis regarding Colic-Associated Dystemperament states that the intestinal microbiome’s chemical signalling may play a role in both causing the inflammation/bloating/pain associated with the colic and the dystemperament that often accompanies the condition. There seems to be consensus that the condition is caused by localized gas buildup and associated bloating, however, there is no consensus beyond this. It is unknown, for instance, why it is that some children experience colic during infancy while others do not and how it is that colicky infants overcome the condition. If so, other questions include whether having had colic carries positive or negative ramifications for health later in life and what the nature of those consequences may be. For instance, appendicitis during the adolescent or adult years may be a consequence of failing to go through the rigors of colic in infancy i.e. appendicitis may be an adult manifestation of the condition known in infancy as colic. As with conditions such as Chicken Pox, the later in life the condition strikes, the stronger the immune response and the more severe the symptoms. An immune component of colic would suitably explain why symptoms are longer in duration but lower in intensity when contracted during infancy and why, if my hypothesis is correct, an initial contracture post-infancy carries far greater risk of appendicitis requiring emergency medical attention. A greater degree of inflammation brought on by a stronger immune response increases the likelihood of the deterioration of cell walls and coherent tissue structures, potentially effecting the rupture of the appendix.

Central to my reasoning on this matter is a chance observation made by this author of a similarity between between Colic-Associated Dystemperament and anecdotal observations reported by many concerning remarkably similar effects upon mood associated with the consumption of brown liquors not seen with clear liquors. These seemingly-unrelated phenomena share in common

that they may be prompted by a combination of inflammation leading to intestinal constriction (in the large colon) as well as modest gas generation leading to relatively small but consequential pockets of bloated colon. When it comes to brown liquors, tannins have been blamed both for hangovers and mood-altering effects sc. dystemperament. Medical professionals have an unfortunate tendency to deny that this difference between brown and clear liquors exists at all. If asked by a patient in a clinical setting, doctors will generally respond by chastising patients for consuming alcohol whatsoever and insisting that, "all alcoholic beverages make people angry." It is not clear whether this tendency has its roots in ignorance or, rather, a fear of being perceived to endorse alcohol consumption by holding one beverage type in higher esteem than another. Virtually no research has been conducted into this phenomenon.

It has been observed that pain and irritability go hand-in-hand, particularly in the case of chronic pain. It has long been assumed that this irritability is a natural emotional response to being in constant pain. The presence of chronic pain can magnify the perceived importance of the smallest insults. It is a fact that a person in physical pain will frequently misdirect their anger at any source of grief, however minor. Somewhere; in a part of the human brain devoid of rationality; an instinct drives people to seek to put a name and a face upon the source chronic physical pain in the hopes that they may counter and neutralize that source of pain so as to prevent further injury. Colic-Associated Dystemperament has long been attributed to an emotional reaction to chronic pain rather than what this author believes is responsible: A direct effect of a nerve signal similar to pain that has singular effects upon neural function which, although it is often sent alongside pain signals, may be conveyed to the brain even in the absence of pain signals. While it may be possible that this phenomenon can be explained by the brain gradually changing the way it handles pain signals, I would posit that the brain signals themselves morph with time due to chemical exhaustion of the mitochondria of the cells that make the electrically-active reflector molecules that, when released, cause electrical signals to be bounced back to the brain, leading to the perception of pain. Metabolic exhaustion of the nerve cells in the case of chronic pain results in shortcuts being taken in their manufacturing process, leading to structural changes to the "reflector molecules" resulting in a return signal that is qualitatively different than a traditional pain signal. These signals electrically cue one to become instantly enraged regardless of the actual duration of the pain. Pain signals originating from these "deformed reflectors," rather than having the effects associated with pain e.g. capturing attention and distracting focus away from other tasks, crying for help and disrupting motor function to prevent further injury, instead, provoke extreme anger.

This is contrary to existing doctrine that holds that either: A.) Irritability associated with intestinal inflammation is emotional or B.) Inflammatory cytokines capable of crossing the blood-brain barrier are released from the inflamed tissues of the large colon and are responsible for brain inflammation leading to irritability. Rather, the correct answer would seem to be C.) *Signals similar to pain signals not interpreted by the individual as pain have an effect on neural function that causes extreme irritability.* This hypothesis would seem to fit well within our understanding of "evolutionary selection" as

sensing “new” pain is useful for avoiding further damage, but can inhibit performance in combat situations e.g. one may need to punch someone in the face with a broken hand, but fear of pain may inhibit the performance of this critical act. Animals may need to flee from a predator by running on an injured leg, something that would surely slow that animal down if it could not “tune out” the pain.

If a pain is perceived by the brain as “old” or recurring pain (gas bloating of the large colon fitting this description in most cases given that people experience this sort of pain repeatedly over the course of many years living,) it may lead to relatively modest amounts of perceived pain with disproportional changes to their temperament. In the case of brown liquors, I would suggest that the disinhibiting effects of alcohol when coupled with intestinal inflammation and gas bloating (driven by ingredients in the liquor,) result in primal, aggressive tendencies being exhibited.

Recent studies have linked aggression in select cases of Autism Spectrum Disorder to intestinal disorders, further supporting my hypothesis. Prior to the recent study on this matter, it was said to be unknown why some children with ASD exhibited violent behavior while others did not. It was speculated (controversially) that the condition of autism, itself, underpinned the violent tendencies. I would suggest that it is the aforementioned quasi-pain signal driving the violence-in-ASD phenomenon and not the ASD, itself. It is the inability of the child with ASD to communicate that they are in pain which causes it not to be remedied and results in the extreme irritability. In short, they are suffering from gas bloating in nearly all cases.

Yet another condition associated with irritability, Pre-Menstrual Syndrome, has been uniformly attributed to hormonal changes for over a half century. Given the possibility of an oblique quasi-pain signal, the basic medical assumptions surrounding PMS need to be reconsidered. The uterus would undergo the same sort of chronic stretching and inflammation the intestines would during gas bloating. *This would explain why it is that the irritability associated with PMS is alleviated by NSAID pain relievers and not other types of pain relievers.*

Conclusion

We must continually re-evaluate our assumptions in scientific matters, particularly in those matters wherein the science is said to be “settled.”